tured with the aperture to the balloon at right angles to the long axis of the tube and at least three inches from the proximal open end intended for suction.

Of probable diagnostic significance is the spherical shape of the mercury, representing a surface tension phenomenon in the presence of another fluid. Mercury alone tends to appear as an amorphous mass. Hence, when mercury appears globular, an attempt should be made to aspirate fluid from the balloon.

SUMMARY

A case of intestinal obstruction due to excess fluid in the balloon of the Miller-Abbott tube re-emphasizes the need for proper bedside care to prevent such a complication. It is also suggested that the two proximal ends of the double lumina be more clearly separated at the time of manufacture.

184 Nemaha Street, Pomona (Hauch).

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Torsion and Infarction of the Normal Ovary in Children

L. GORDON FISKE, M.D., Santa Barbara

AN UNUSUAL CAUSE of acute abdominal symptoms in children is torsion with infarction of a normal ovary. Hinshaw and Kugel[4] reported such a case in 1956 and said that 21 cases in all had been reported in the English literature up to that time. However, Baron[1] in 1932 reported a series of 26 cases. Downer and Brines[3] collected reports of 18 cases from the literature and reported a case they had observed. Many of the cases reported in each of these series are of course also included in the others. Baron reported one instance of torsion of the normal ovary, and later torsion and infarction of the remaining ovary in the same patient, necessitating bilateral oophorectomy and salpingectomy.

Although such a condition should possibly be considered in the differential diagnosis of lower abdominal pain, its rarity would put it far down on the list of possibilities. Torsion of the right ovary almost invariably causes clinical symptoms that may be mistaken for those of acute appendicitis, as happened in the case herein reported. Torsion is more common on the right side than on the left. The diagnosis is difficult because of the rarity of tubo-ovarian disease in children. The symptoms of ovarian torsion may simulate those of bowel obstruction or ureteral colic. Rarely is the diagnosis made until exploratory laparotomy is done because of the obviously acute abdominal emergency.

In cases reported previously the clinical features have been similar. At first the pain is cramp-like and not too well localized. Vomiting frequently ensues, and the pain then localizes to either of the lower quadrants of the abdomen and becomes more steady in character. Sometimes a history of previous attacks can be elicited. Upon physical examination, tenderness usually is noted either on the right or left side of the lower abdomen and in some cases other signs of peritoneal irritation are present. There may be radiation of pain to the thigh or genitalia. In several reported cases it was noted that a small, tender pelvic mass was felt on rectal or pelvic examination.

REPORT OF A CASE

A 12-year-old white girl was taken to a physician's office in the early morning of April 28, 1957, because of abdominal pain which had begun 12 hours previously. At first the pain was described as being cramp-like, and not localized. By the time of first examination the pain was beginning to localize to the right lower quadrant and was becoming more steady in character, but with occasional exacerbations. Mild tenderness was noted in the right lower quadrant, but there was no rebound and no rigidity. No masses were felt either abdominally or rectally. The patient did not have fever. Leukocytes numbered 5,000 per cu. mm. and the cell differential was within normal range. No abnormality was noted in the urine.

The family was instructed to bring the child back later in the day for further evaluation, and it was in the late afternoon on her return that surgical consultation was obtained. During the day the pain had become increasingly severe and steady and was more decidedly localized to the right lower quadrant. Vomiting had occurred during the day. The patient had no previous history of digestive distress or symptoms referable to the genitourinary tract. She had had two menstrual periods, the second one having been a week before the present illness. Upon examination the patient appeared to be acutely ill. She tended to lie on her right side with the legs flexed. The temperature was 99.6° F., the pulse rate 100 and respirations 22 per minute. The face was pallid and the skin clammy. There was pronounced right lower quadrant tenderness below McBurney's point, with guarding and mild rigidity. Peristalsis was hypoactive. Upon rectal examination tenderness was

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noted greater on the right, but no masses were felt. The hymen was intact. The impression was very definitely that the patient had acute appendicitis, and she was immediately admitted to the hospital and prepared for operation. At the time of admittance, leukocytes numbered 10,900 per cu. mm.—79 per cent polymorphonuclear cells, 1 per cent eosinophils and 20 per cent lymphocytes. The hemoglobin value and the erythrocyte content were within normal limits. No abnormality was noted in the urine.

A McBurney incision was made. The appendix appeared to be normal but there was a small amount of clear, straw-colored fluid in the lower abdomen. A mass was felt in the cul-de-sac and upon examination it was observed to be the right tube and ovary. The ovary was enlarged to approximately 7x4x3 cm. in diameter. It was dark and edematous and the surface mottled with extravasated black clotted blood. The tube was purple, swollen and edematous. The pedicle was unusually long and was twisted for two complete turns at its base. Immediate resection was considered, but first the pedicle was untwisted and the ovary and tube replaced in the abdomen while the appendix was being removed. Several minutes later they were inspected again, and there was then obviously a return of arterial circulation. The tube was bright red and the ovary, which had diminished in size by about one-third, had taken on a pinkish-white coloration except for the small areas of hemorrhage on the surface. When the surface was nicked in several places, bleeding showed that there had been a return of adequate arterial circulation. It was decided to preserve the ovary and the tubo-ovarian structures were sutured in normal position to the right broad ligament in such a way that torsion could not recur. No complications developed and the patient was discharged from the hospital on the third postoperative day.

COMMENT

Some observers have ascribed torsion of the ovary to unusual length of the pedicle, as in the case herein reported. However, in the case reported by Baron, in which torsion of first one and then the other ovary occurred, the second ovary was noted to have a normal looking pedicle at the time of the first operation. Torsion may be repeated, causing intermittent or recurrent attacks as in the case reported by Hinshaw and Kugel.[4] Torsion causes circulatory stasis, venous occlusion at first, then interruption of arterial flow as torsion progresses. Hemorrhagic infarction finally results.

In ovarian torsion, the right ovary twists clockwise and the left ovary counterclockwise, as viewed with the pedicle pointing away from the observer (Küstner's Law).

It is never possible to state with complete assurance that the adnexae were normal before twisting occurred, because the extensive hemorrhage, necrosis and infarction which follow the twisting would obliterate evidence of any mild inflammation that might be present. Downer and Brines,[3] in reporting upon 19 cases, included only those in which the patient was less than 16 years of age, expressing the thought that under that age venereal disease probably would not be a considerable factor. This limit is doubtless too high, and setting the age limit at 13 years would have reduced the number of cases from 19 to 12—nine in which there was torsion of ovary and tube, two of the ovary only and one of the oviduct only.

Immediate laparotomy with resection of the involved ovary and tube was the method of treatment in other cases reported. In the present case, however, this was deemed unnecessary after a return of circulation was observed when the pedicle was untwisted. It might be well in other cases, therefore, to wait to see if arterial circulation will return, lest the ovary be removed unnecessarily. Since the earlier part of the process is intense venous engorgement, some damage to the ovary may result but if arterial supply is not blocked by thrombosis, a functioning ovary may be preserved. It would seem that in very young girls, ovarian tissue should always be preserved on both sides if feasible, for there is always the possibility that the function of the remaining ovary could be lost later through some disease process.

SUMMARY

Torsion and infarction of the normal ovary and tube is a rare cause of acute abdominal symptoms in children.

The clinical symptoms and signs, if the right uterine adnexa is involved, resemble those of acute appendicitis. Bowel obstruction or ureteral colic may be simulated. Torsion is more common on the right side than on the left. Since left-sided torsion produces a confusing clinical picture not likely to be mistaken for appendicitis, surgical interference is more like to be delayed.

A case of right-sided torsion with early venous infarction is reported. In this case, unlike others previously reported, the ovary and tube were preserved when arterial circulation was observed to be reestablished soon after the pedicle was untwisted. It is suggested that before oophorectomy is done in cases of ovarian torsion untwisting should be done to permit evaluation of arterial sufficiency. Unless irrevocable arterial block has occurred, an otherwise normally functioning ovary may be preserved.

22 West Micheltorena Street, Santa Barbara.

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Endocarditis Following Ventricular Septal Defect Repair

WILLIAM MANDEL, *M.D.*, San Francisco,
M. S. HOFFMAN, *M.D.*, G. H. HEITZMAN, *M.D.*,
and I. KASS, *M.D.*, Denver

THE INCIDENCE of endocarditis following operations for repair of congenital or acquired cardiac lesions has been stated to be one per cent.[6] Reports of 30 such cases were found in a review of the literature,[1-6,9-11] although in three of the cases proof of endocarditis was lacking. Herewith is the first reported occurrence of endocarditis following repair of a ventricular septal defect. It is the only known case of endocarditis following approximately 1,200 cardiac operations performed in four hospitals in Denver.

REPORT OF A CASE

A 9-year-old boy was admitted to the National Jewish Hospital in Denver on January 10, 1956, for correction of a ventricular septal defect. He had been in good health until, two years before admission, a febrile illness associated with aching in the joints developed. A cardiac murmur was heard at the time. Several weeks after recovery, the patient noted that he was tired and short of breath oftener than before and could no longer keep pace with his playmates. Questioning brought denial of cough, epistaxis, chest pain, hemoptysis, wheezing, paroxysmal nocturnal dyspnea, ankle edema and cyanosis. The patient was not subject to frequent respiratory infections or sore throats. There was no familial history of congenital cardiac defects.

On physical examination, the patient was observed to be thin and well developed and in no acute distress. The blood pressure was 110/60 mm. of mercury and the pulse rate was 100 per minute. Except for those referable to the cardiovascular system, no abnormalities were noted. There was no clinical evidence of increased venous pressure. The left side of the chest was more prominent than the right and showed active cardiac pulsations. Upon palpation of the precordial region a right parasternal heave and a left ventricular apical thrust were noted. The heart was enlarged to percussion. A systolic thrill was felt over the precordial area, being loudest at the third left interspace near the sternum. The murmur was louder in the fourth than in the first intercostal space. The pulmonic second sound was duplicated and accentuated. The femoral pulsations were felt and there was no edema or cyanosis or clubbing of the extremities.

Results of examination of the blood and the urine were within normal limits. Blood cultures were negative for pathogenic organisms. An electrocardiogram showed evidence of left ventricular hypertrophy, which was confirmed by roentgenographic and fluoroscopic examination. Fluoroscopic examination showed enlargement of the main and branch pulmonary arteries and increased vascularity of the pulmonary parenchyma. Cardiac catheterization was carried out and the results were consistent with the diagnosis of a ventricular septal defect. Blood oxygen saturation was increased three volumes per cent as the catheter passed from the right atrium to the right ventricle. This increase was maintained in the main pulmonary artery. The pressure in the right ventricle was 74/0 mm. of mercury and in the pulmonary artery 72/20 mm. of mercury.

On January 20, by means of hypothermia and coronary artery perfusion, a large ventricular septal defect was repaired under direct vision. The immediate postoperative course was satisfactory, and the patient was ambulatory by the fifth postoperative day. The systolic murmur present before operation was no longer audible. However, on the tenth postoperative day a diastolic murmur, characteristic of that occurring in aortic insufficiency, was heard along the left sternal border. Comparison of a chest roentgenogram with previous films showed a slight increase in the transverse diameter of the heart. Although the patient appeared clinically well, fever that varied between 99° and 100°F. daily developed.

Following the operation, the patient was given 600,000 units of procaine penicillin and 1.0 gram of tetracycline daily. The low grade fever persisted, and on the 24th postoperative day the temperature suddenly rose to a range between 101° and 103°F. This was associated with shaking chills and sweats. Blood cultures grew coagulase-positive staphylococcus aureus sensitive to penicillin, erythromycin and chloramphenicol. Although the antimicrobial therapy was changed to 4,000,000 units of penicillin and 1.0 gram of erythromycin daily, fever persisted. Symptoms and signs of congestive heart failure developed on the thirty-third postoperative day. The patient was treated with digitalis, mercurial diuretics, oxygen, increasing doses of penicillin up to 20,000,000 units daily and chloramphenicol. The blood cultures were negative for organisms after the thirty-seventh postoperative day, but the congestive failure progressed. Dyspnea increased and the patient died on the fifty-sixth postoperative day.

Pathologist's report: At necropsy, passive congestion of all organs, most severe in the liver and lungs, was noted. The subcutaneous tissues were edematous. A loculated right hydrothorax was present. The pericardium and epicardium were thickened and the intervening space was largely obliterated by fibrous adhesions. The heart weighed 385 grams. The right ventricular wall was 8.0 mm. thick; the left ventricle was moderately hyper-

From the Department of Cardiovascular Surgery of the National Jewish Hospital, and the University of Colorado School of Medicine, Denver 6, Colorado.

San Francisco Hospital, San Francisco 10 (Mandel).